7 POPULATION REGULATION IN ANIMALS

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'. . . and yet in the long run the forces are so nicely balanced, that the face of nature remains uniform for long periods of time . . .'

CHARLES DARWIN,
The Origin of Species, 1859
Chapter 3

INTRODUCTION

In 1798 the Rev. T.R. Malthus, in his essay on population growth, pointed out that whereas animal populations can increase geometrically, their food supplies usually increase arithmetically; and consequently animal populations always increase up to the limit of their resources. Mortality from lack of food through starvation and from disease then acts to prevent further increase. It was not until Charles Darwin read this essay that he understood the significance of intraspecific competition for resources for his theory of Natural Selection. The Malthusian hypothesis has been at the centre of all subsequent debate on what regulates populations. Before reviewing the history of this debate it is necessary to explain the meaning of the terms commonly used.

LIMITATION, REGULATION AND PERSISTENCE

I shall use the term 'population' to mean a group of co-existing individuals which interbreed if they are sexually reproductive. Some populations frequently go extinct (e.g. bird populations on islands, Diamond 1969) and are then re-established through founding immigrants. Many other populations have remained extant for long periods and show no signs of becoming extinct. It is this 'persistence' of populations which has led some of the earlier population ecologists to suggest that there is a demographic mechanism which at lower density prevents extinction and at high density provides an upper limit to population numbers in concordance with the Malthusian principle.

Populations have inputs of births and immigrants (production) and outputs of deaths and emigrants (losses). Figure 7.1 expresses the

processes of production and loss as a percentage of population size N. For simplicity, production P is held constant with density. In temporal sequence a constant mortality (m_1), say at an early age, reduces this production [Fig. 7.1(a)]. There follows a mortality (m_2) which increases linearly as population increases. This percentage increase in mortality (or

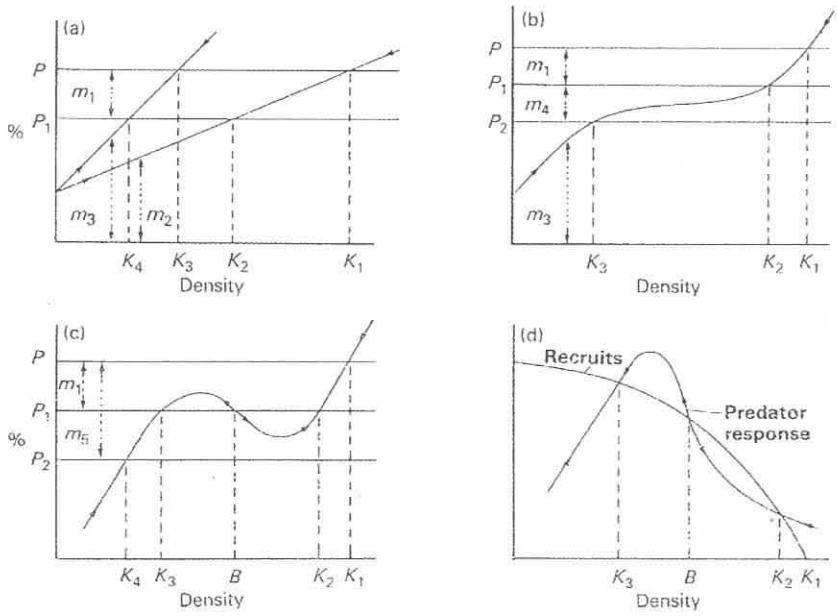

FIG. 7.1 Model of density-dependent and density-independent processes,
(a) Production (P), is held constant over all densities. In sequence a density-independent mortality, DI, (m_1) reduces production (P_1). There follows a density-dependent mortality, DD (m_2 or m_3). The intersect of production with density-dependent mortality determines the equilibrium densities ($K_1 - K_4$) [developed from Krebs (1985), after Enright (1976).]
(b) Density-dependent mortality (m_3) is curvilinear. Both m_1 and m_4 are density independent and reduce production by the same amount. A constant DI reduces the equilibrium to a smaller degree (K_1 to K_2) when DD is stronger, than when DD is weaker (K_2 to K_3).
(c) There are two ranges of density with density-dependent mortality and an intermediate range when there is inverse density dependence. A density-independent mortality, m_1, results in two stable equilibria, K_2, K_3 and an unstable equilibrium at boundary, B. A larger DI (m_5) results in only one lower equilibrium K_4.
(d) Production reduced by density-dependent mortality through lack of resources (as mimicked by the logistic equation) produces the (recruits) curve with equilibrium K_1. The predator total-response curve intersects at stable equilibria K_2, K_3 and at boundary point B.

equivalent percentage drop in production) with population increase is termed *density dependent* and the causes of this relationship are called *density-dependent factors*. Conversely, processes which show no relation to population size (being either constant or random, such as m_1), are termed *density independent* and their causes *density-independent factors*. In addition the reverse relationship to *density dependence* is termed *inverse density dependence*. Where m_2 intersects P_1 there is a stable equilibrium point K_2 because losses balance production remaining after the initial mortalities. Any temporary perturbation of the population above K_2 induces the mortality m_2 to be higher than P_1 and the population returns to K_2. If for some reason the density-independent mortality (m_1) is not imposed then the stable point moves to a higher population size K_1. If the intensity of the density-dependent mortality is strengthened (m_3) the slope increases and the population decreases to K_4.

Several points emerge from Fig. 7.1(a):

(i) The position of the equilibrium point is set by both density-dependent and density-independent factors. The process which sets the equilibrium point is termed *limitation* and the factors which cause the changes in production or loss are *limiting factors*. It is obvious that *any* factor causing a change in production or loss is a limiting factor. Therefore, it is a trivial question to ask whether a particular mortality factor limits a population. The more interesting questions ask: (i) to what extent; and (ii) how does a particular factor change the equilibrium position?

(ii) The process whereby a population returns to its equilibrium is termed *regulation* and the factors causing this are *regulating factors* (e.g. Nicholson 1933; Murdoch 1970). Such factors must have a density-dependent effect.

(iii) A change in the density-independent mortality causes a greater change in equilibrium (K_1 to K_2) when the density-dependent mortality is weak (lower slope) than when the latter mortality is strong (K_3 to K_4).

(iv) A change in the strength (slope) of the density-dependent process alters the equilibrium set point to a greater extent when the mortality acts earlier in an animal's life and before much of the density-independent mortality (K_1 to K_3) than when density dependence occurs later (K_2 to K_4).

Figure 7.1(a) is a simple model in which it is assumed that production is constant; the conclusions do not alter if production is also density dependent. Furthermore, in Fig 7.1(a) I have assumed that density dependence is linear over all values of N. There is no *a priori* justification for this and density dependence could be curvilinear or even take the

complex form of mortality (m_3) shown in Fig. 7.1(b). Here there are two regions of N with strong density dependence and a middle region where density dependence is weak. It follows from the points discussed above that in this model, the same degree of density-independent mortality (m_1 or m_4) will have greater effect on altering the equilibrium point if it is in the middle region of N (K_2 to K_3) than at either end (e.g. K_1 to K_2).

Figure 7.1(c) develops the sequence of models so that the population now experiences an inverse density-dependent mortality at intermediate values of N. Imposition of a density-independent mortality m_1, results in there being two stable equilibria K_2 and K_3 and an unstable boundary point B. If the density-independent mortality is larger (m_5) then only one, lower stable point K_4 exists. This kind of scenario could arise if a prey population was regulated at low densities by predators and at high densities by intraspecific competition for resources. In fact Fig. 7.1(c) does not differ from the familiar Rosenzweig–MacArthur models (e.g. Ricklefs 1979, p. 618) for prey recruitment and predator total response curves [Fig. 7.1(d)].

Figure 7.1 illustrates the difference between limitation and regulation. It is clear from the literature that these two terms as well as the term 'control' have been used interchangeably and one has to infer, if possible, from the context, what meaning is intended. Often one finds density-dependent regulation which is a tautology, and density-independent regulation which is a contradiction — it should be density-independent persistence. I shall confine the use of control to the context of biological control where one wants to depress a pest population to some arbitrary but economically suitable low level (Milne 1957).

HISTORY OF CONCEPTS

The balance of nature

Nicholson (1933) revolutionized thinking about population limitation when he introduced the concept of regulation through density-dependent factors. This idea was stimulated by the observation that populations rarely went extinct. His premise was that persistence would occur only if some form of governor was acting on the population. Thus, in his own words (Nicholson 1933) 'Populations must be in a state of balance with their environments' (p. 176); 'For the production of balance, it is essential that a controlling factor should act more severely against an average individual when the density of animals is high, and less severely when the density is low' (p. 135); '[that is] it is essential that the actions of a controlling factor should be governed by the density of the population

controlled, and competition seems to be the only factor that can be governed in this way . . ., generally competition between animals when seeking the things they require for existence, or competition between natural enemies that hunt for them' (p. 176).

Factors which are governed in their action by the density of the population were more aptly described as density dependent by Smith (1935), and although Nicholson persisted in using his own more cumbersome terminology, Smith's terms have now become the common usage. Smith developed his terms from two economic entomologists Howard & Fiske (1911).

Nicholson reiterated his ideas but they remained essentially unchanged (Nicholson, 1954a, b, 1958; Nicholson & Bailey 1935) and were accepted more or less by Varley (1947), Elton (1949), Solomon (1949) and Lack (1954, 1955). The main points are: (i) that the environment is composed of density-dependent and density independent factors; (ii) that regulating factors must be density-dependent, the chief of which is intraspecific competition for resources, but others such as predators and parasites can also be density dependent; (iii) there can be more than one density-dependent factor some of which act together whilst some can compensate for each other; (iv) density dependence may not operate all the time — there are times of increase and times when density-independent factors cause a decrease without compensating effects from the density-dependent factors (Nicholson 1958, p.168); (v) scramble (exploitation) competition can cause wastage or spoilage of resources with the result that populations overshoot the equilibrium and cycles occur, whereas contest (interference) competition causes less wastage and greater dampening of cycles; and (vi) parasite–host interactions in insects are unstable and can cause local extinction because parasites act in a delayed density-dependent way (Varley 1947). However, since in nature populations live in a patchy environment, patches can be recolonized by emigrants from other patches. In particular, interference competition (e.g. territoriality) promotes the production of emigrants which act to regulate the home population while recolonizing other areas (Nicholson 1958, p. 169).

This theory came under heavy criticism in the 1950s. Some of the main criticisms were: (i) the logical necessity for density dependence is a mathematical result of the premise that there is an equilibrium. If this premise is false then the rest of the theory collapses and it is unlikely to be applicable in nature (Andrewartha 1958; Milne 1958); (ii) Andrewartha & Birch (1954, p.19) stated '. . . that density-independent factors do not exist . . . [so] there is no need to attach special importance to density-dependent factors . . .'; and (iii) Milne (1957, p.203) considered that

'. . . numbers cannot stop falling unless the density independent environment is or becomes favourable for that purpose. The ultimate control of fall in numbers rests, therefore, with the density independent factors'.

The first of these criticisms is fair enough. The critics claimed that since there is no obvious balance in nature, there is no need to dream up a mechanism for it. By the same argument, however, there is no reason why regulation should not actually exist. The test to distinguish between the two hypotheses is to measure density dependence in the field and predict the population equilibrium. Varley (1947) was the first to attempt this in his studies of parasitism of the knapweed gallfly, but Milne (1957, p. 199) countered that the data did not support the process of density dependence. By 1958 therefore, 25 years after the theory of population regulation had been proposed, there was little if any incontrovertible evidence in nature in its favour. Indeed, Andrewartha (1958) considered it was untestable. Both of the other two criticisms above reflect a misunderstanding of how regulating factors work.

Weather and population persistence

Early theories (e.g. Uvarov 1931) proposed that weather alone limited animal populations. These were superseded by the theory of Thompson (1929, 1939) who considered that '. . . populations are not self-governing systems.' They are limited by a variety of factors; occasionally all factors favour a population which can then 'outbreak' by rapid increase. For the most part, however, one or other factor keeps the population down. Thompson makes no reference to density dependence except for the enigmatic remark (1939) that '. . . the Universe is a density-dependent factor'. Thompson, himself regarded his theory as philosophical, even metaphysical. Milne (1957) agreed that it was '. . . certainly subtle and elusive'. One is left with little idea of how to test such a theory and the uncomfortable feeling that it may not be testable.

Andrewartha & Birch (1954), Birch (1958) and Andrewartha (1958, 1961) advanced the idea of environmental limiting factors, and as mentioned above, did not consider the environment could be divided into density-dependent and density-independent factors. In their words:

> The numbers of animals in a natural population may be limited in three ways: (a) by shortage of material resources, such as food, places in which to make nests, etc.; (b) by inaccessibility of these material resources relative to the animals' capacities for dispersal and searching; and (c) by shortage of time when the rate of increase, r, is positive. Of these three ways, the first is probably the least, and the last is probably the most, important in nature. Concerning (c), the fluctuations in the value of r may be caused by weather, predators, or any other component of environment which influences the rate of increase.

In essence (a) and (b) are the same — populations can be limited by resources but this rarely happens because other factors, notably weather, impose their effect before resources run out. Their idea is effectively the same as that of Thompson. Their work was conducted largely in Australia where both weather and populations fluctuate markedly; they were concerned with explaining these fluctuations in numbers. Two of their classic studies were on the grasshopper *Austroicetes cruciata* and the rose thrips *Thrips imaginis*. Populations are divided into sub-populations; those in the centre of the species' range are limited by many factors, including migration of dispersers. Populations on the edge of the range experience harsh climate and these populations may go extinct, later to be recolonized. Thus Birch (1958, p. 206) wrote 'We have maintained that there is a chance of extinction for the grasshopper in any year in any one place . . . but the chance of extinction in all places in any one year is infinitesimally small under present climatic conditions.'

It is evident that Andrewartha and Birch were concerned with limitation and limiting factors, and their conclusions concerning these are quite correct. Their disagreement with Nicholson was unnecessary because they were arguing at cross purposes — Nicholson was concerned primarily with regulation. Indeed, Nicholson's view of limitation (see above) does not differ substantially from that of Andrewartha & Birch. Another distinction was that Nicholson was concerned with populations in moderate and stable environments near their equilibrium, whereas Andrewartha and Birch examined those not near their equilibrium and in fluctuating environments.

However, the most contentious aspect of Andrewartha & Birch's theory is that populations could persist without regulation. It is generally recognized that such populations would show a random walk through time and would eventually go extinct; hence Elton's (1955) review of their book stated, 'I cannot believe that the natural communities of the world will be found to have evolved solely by guess and by chaos.'

Milne (1957, 1958) reviewed the various theories, and finding none to his liking, came up with a modified version of both Nicholson's and Andrewartha & Birch's ideas. He claimed that first, intraspecific competition is the only perfectly density-dependent factor and thus operates only at high density, but rarely; second, populations are usually held at low levels by a combination of density-independent and imperfect density-dependent factors such as predators and parasites; third, since density-dependent factors cannot stop a population declining to extinction, density-independent factors must do so. Milne considered the idea of imperfect density dependence as his contribution, but in fact

Nicholson had already suggested it. Milne's third point reflects a confusion between the actions of limiting and regulating factors and is illogical (Solomon 1959).

This covers the main theories and some of the important criticisms up to the late 1950s. Peculiarly, they centred on insects in Australia. Nicholson's view had developed from theory, was more complete and more robust, but apart from some early work by Reynoldson (see Reynoldson 1966 for review), there was little hard field evidence in its favour. Andrewartha & Birch based their ideas on extensive field measurements but the theoretical development was incomplete and unsatisfactory.

Extrinsic factors, intrinsic factors and group selection

By 1960 Nicholson's views were the more popular and there was a burst of field studies to test them. In so doing new debate arose concerning the cause and mechanism of regulation. Debate shifted to Britain and centred largely on bird studies.

Lack (1954, 1966), as the leading spokesman for the Nicholson view, proposed that extrinsic factors — factors external to the organism such as resources, predators and parasites — acted to regulate populations. Summarizing the specific mechanism Lack (1966, p.290) argued that '. . . in birds the reproductive rate of each species, evolved through natural selection, is that which results in the greatest number of surviving offspring per pair, and that the population density is regulated by density dependent mortality, in most species by food shortage outside of the breeding season'. He conceded that other species, particularly phytophagous insects, could be regulated by predators. He based his ideas on the long term studies of great tits (*Parus major*) at Wytham Wood (Perrins 1965) and other studies, some of which are collated in his 1966 book. The two essential elements of his idea are first, that the density dependence occurs outside the breeding season, and second, that behavioural attributes of the animals do not result in regulation. In particular, territoriality does not regulate numbers but merely determines who is able to breed.

This view was in stark contrast to a substantially new theory that was developing. It proposed that intrinsic factors,those characteristics of the individual such as behaviour, physiology and genes, could act to regulate the population at a level below that imposed by the food supply. This is the 'self-regulation hypothesis'. Chitty (1960), in an important early paper, suggested that the decline phase of vole (*Microtus agrestis*) population cycles occurred as a consequences of intraspecific aggression

during periods of high density. This aggression caused voles to become susceptible to a variety of secondary environmental factors, mortality ensued and the population declined. Chitty (1967) developed this idea into the 'Genetic hypothesis', (Krebs 1978) in which he suggested that the population is genetically polymorphic for aggression. A non-aggressive high reproducing morph is favoured by natural selection at low and increasing densities, but at high densities a large, aggressive but low reproducing morph is favoured. Because of both low reproduction and aggression the population then declines. Although Chitty considered that aggression is selected for ultimately through competition for food, proximately it is the degree of social interaction to which the individual now responds as the population no longer experiences food limitation. Recently, Chitty (1987) has modified the mechanism of social interaction but maintains the concept of a genetic polymorphism.

A major conceptual problem with the genetic hypothesis is how a population becomes emancipated from its food supply. If aggressive behaviour has evolved through natural selection by intraspecific competition, then the effects of food limitation must still be present to maintain the behaviour in the population (i.e. the population remains food limited). If food limitation does not occur, then it would appear that a mutant individual which could breed at high density would be selected for and the population would increase until it is again limited by food. So far this problem has not been resolved. However the hypothesis has stimulated many studies to test the behavioural (Myers & Krebs 1971; Krebs *et al.* 1973; Lidicker 1975; Tamarin 1980) and genetic aspects (Krebs & Myers 1974; Tamarin 1978). Two significant developments were C.J. Krebs' experiments demonstrating the 'fence effect' — the radical change in population when dispersal is inhibited (Krebs *et al.* 1973) and Lidicker's (1975) proposal of regulation through density-dependent dispersal. I will return to the behavioural aspects later, but the genetic evidence in favour of the idea remains inconclusive (McGovern & Tracy 1981). A recent test of heritability of Boonstra & Boag (1987) indicated little heritability.

While Chitty's ideas were gaining popularity, Christian suggested mechanisms for how aggression could affect the animal (Christian 1961; Christian & Davis 1964). Endocrine responses to increasing density affected reproduction primarily through: (i) aggression, (ii) stimulation of adrenal function and production of steroids; and (iii) a sensitivity of the juvenile hypothalamus. These, Christian (1980) suggests, are density dependent, although details of the mechanisms remain unknown. This work has been largely confined to mammals and to what extent it applies to other groups of animals is unclear. However the general idea of

Chitty's genetic theory of population regulation was readily applied to other classes of animals: Pimental (1968) used it to interpret changes in insect populations.

In 1962, Wynne-Edwards (1962, 1965, 1986) proposed his theory of regulation through behaviour specifically functioning to keep the population below the level determined by food supply. He stated (1965) 'It is readily apparent that, although food is generally the commodity that ultimately limits the carrying capacity of the habitat, the population dependent on it must not be allowed simply to increase until further growth in numbers is chronically held in check by general starvation. Famine is a catastrophe especially likely to damage the resource and lead to its permanent depletion'. Two necessary conditions follow from this proposition: (i) the mechanism of regulation operates to limit reproduction usually by behavioural means so that mortality from starvation is avoided (he concedes some mortality through stress could occur); and (ii) such regulatory mechanisms evolve through 'group selection'.

Group selection (or interdemic selection) requires that: (i) there is effectively no genetic interchange between groups or demes; and (ii) groups which overexploit their resources go extinct. Thus a gene that causes animals to limit reproduction so as to keep a population lower than the limit set by food would prevent demes from going extinct, so eventually only demes with this gene would survive.

This theory of Wynne-Edwards caused controversy. Its predictions were the opposite to those of Lack's (1966) theory. Wynne-Edwards considered reproduction was regulated through behaviour such as territoriality, dominance, aggression, inhibition of breeding and dispersal, while Lack proposed regulation through mortality in the non-breeding season, with behaviour playing little or no part. Such a juxtaposition of ideas naturally stimulated an explosion of research in population dynamics on the one hand, and behavioural mechanisms of regulation and their evolution on the other (for example see the symposia edited by Watson 1970; Ebling & Stoddart 1978; Alexander & Tinkle 1981; Sibly & Smith 1985). This work produced much evidence for density dependence from population studies and a plethora of behavioural mechanisms which potentially could regulate populations, though regulation was rarely demonstrated (see below). Group selection and the evolution of 'self-regulation' has been severely criticized on theoretical grounds and it is now generally discounted (Maynard Smith 1964, 1976; Wiens 1966; Bell 1987). There are a few special cases where group selection might occur (Wilson 1983; Harvey 1985) but they differ from the type of group selection promoted by Wynne-Edwards. On the whole observations do

not support the two conditions necessary for group selection to operate. Wynne-Edwards' hypothesis rests on the assumption that populations regulated by resources, damage those resources (i.e overexploit them) and so decline in numbers. There is no *a priori* reason why this should be so and there is now plenty of evidence to show the assumption is false.

REGULATION AND TESTING FOR DENSITY DEPENDENCE

One of the major problems in determining whether or not regulation occurs, has been the difficulty of distinguishing between non-regulated and regulated populations. The most robust evidence comes from perturbation experiments; populations are artifically reduced from or increased above their starting densities. If they return to the level of the unaltered control population, then this is evidence for regulation and such convergence is evidence for density dependence (Murdoch 1970). These experiments are rarely conducted.

The most common approach involves the statistical analysis of census data. The earlier approaches (Morris 1963; Solomon 1964) used a modified Ricker (1954) curve. A plot of log population numbers in one year (N_{t+1}) against log numbers in the year before (N_t) with a slope less than one, if it is not due to some change in weather, could be interpreted as showing density dependence. Morris (1963) provides the classic example with his spruce budworm plot (Fig. 7.2). However this approach has been criticized on the statistical grounds that it can show spurious density independence (Maelzer 1970; St. Amant 1970).

A more robust approach was that of Varley & Gradwell (1960, 1968). The mortality is expressed as log (N_t/N_s) (called the k-value) where N_t is the original population density and N_s is the density of survivors. The k-values are plotted against log N_t, and with appropriate statistical precautions, a positive slope indicates density-dependence. Thus

$$k\text{-value} = \log (N_t/N_s) = \log a + b \log N_t \qquad (1)$$

where the slope 'b' is a measure of the strength of density dependence. This model is linear and it does not always fit the data. Other models have been developed which in logarithmic form are curvilinear (e.g. May *et al.* 1974), and perhaps the best of these is that of Hassell (1975):

$$\log (N_t/N_s) = b \log (1 + a N_t). \qquad (2)$$

This model has the advantage that the k-value plotted against log N_t is

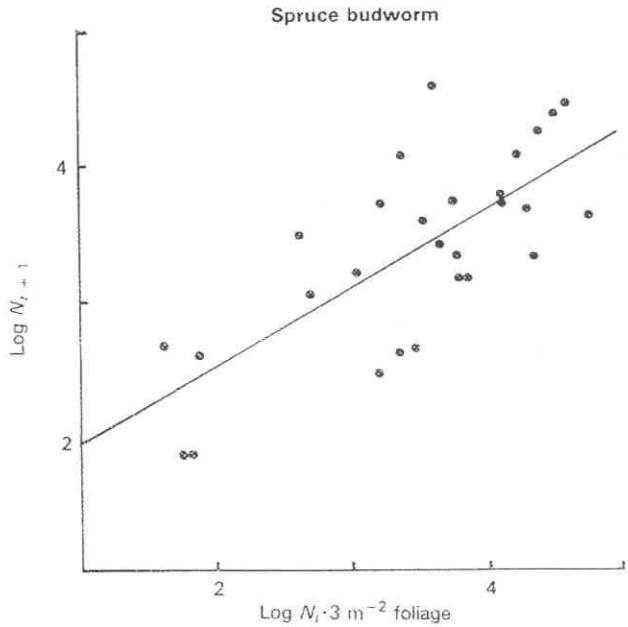

FIG. 7.2 Log density of spruce budworm (*Choristoneura fumiferana*) in each generation (t) plotted against log density in the following generation ($t + 1$). [After Morris (1963).]

nearly linear at high densities. It describes density-dependent relationships from field data reasonably well (Fig. 7.3).

The k-value approach of Varley & Gradwell (1968) and related models (Hassell 1975) have been criticized for being too conservative — they miss too much of the density dependence (Slade 1977; Vickery & Nudds 1984). The autoregression method of Bulmer (1975) is effective only when the population is stationary, while the method of Slade (1977) requires the population to be changing. Vickery & Nudds (1984) suggest another method using a simulation regression test. Recently Gaston & Lawton (1987) have tested these alternative methods using census data from natural populations known from independent evidence to be subject to density-dependent processes. All methods failed to detect density dependence reliably, irrespective of sample size and the dynamics of the population. They concluded that none of the methods was a useful test of density dependence in sequential censuses. These results strengthen the argument for returning to well-designed perturbation experiments.

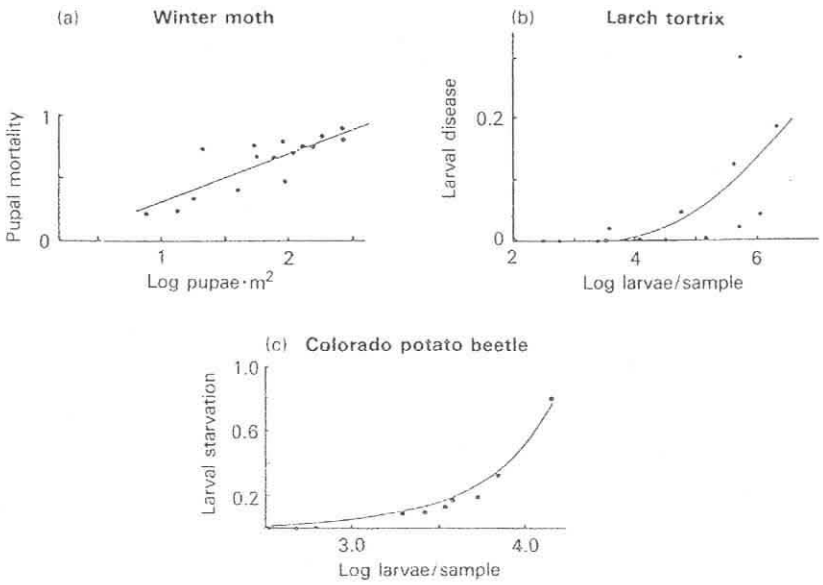

FIG. 7.3 Examples of density dependence in insects from field data, described by equation (2) in text. All are log (N_t/N_s) against log N_t.
(a) Winter moth (*Operophtera brumata*) pupal mortality. [After Varley & Gradwell in Hassell (1975).]
(b) Larch tortrix (*Zeiraphera diniana*) larval disease. [After Varley & Gradwell in Hassell (1975).]
(c) Colorado potato beetle (*Leptinotarsa decemlineata*) larval starvation. [After Harcourt in Hassell (1975).]

Despite all this, most workers are still using the Varley & Gradwell k-value approach. Dempster (1983) used this method to review a number of Lepidopteran populations. He concluded that in one third of the cases no density dependence could be found. Hassell (1985, 1987) challenged this by arguing that life-table studies using average census data may hide density dependence by predators reacting to spatial patchiness in host density or other forms of within-generation heterogeneity. Thus in viburnum whitefly (*Aleurotrachelus jelinekii*) inter-generation density dependence was not detected (Southwood & Reader 1976) because the important density dependence operates from leaf to leaf within a viburnum bush (Hassell, Southwood & Reader 1987). Jones *et al.* (1987) also found that density-dependent mortality of *Pieris rapae* was spatially and temporally patchy.

Murray (1982) has resurrected an old argument of Andrewartha & Birch (1954) claiming that in a territorial species the number of

territories can determine population size without the action of density-dependent factors; and like the earlier arguments he has confused limitation with regulation. Territorial space limits the population but competition for that space provides the regulation and is a function of population size, as is explicitly demonstrated in his own model. One other problem is that he defines the density-dependent process strictly as that in Fig. 7.1(a), with uniform effect over all densities; and if it is not uniform then it is not density dependent. His conclusion follows from his definition, but the latter is arbitrary and not one that is generally accepted.

It is now more than 30 years since the 1957 Cold Spring Harbor Symposium on population studies when there was no known way to detect regulation in the field. The intervening period has shown a progressive sophistication in measuring density dependence through statistical means, positions are less rigid in the debates and we are more aware of the pitfalls in measurement. Nevertheless we are likely to be missing much of the density dependence in the data and some methods may be providing spurious answers (Royama 1977). We should proceed with caution.

EVIDENCE FOR REGULATION

Referring to the statistical problems of measuring density dependence, Murdoch (1970) commented 'It is in these circumstances that the beauty of the convergence experiment as a test for regulation becomes apparent'. Yet there are still very few examples of this type of manipulation. Because of logistical difficulties, most are incomplete or unbalanced. For example, a population of elk (*Cervus elephus*) in Yellowstone National Park, USA was reduced from around 15 000 in 1933 to 4000 animals in 1968 and then left alone (Fig. 7.4): It returned to 12 000 in a few years and is now levelling out at about 18 000 (D. B. Houston 1982 and personal communication). Regulation, it seems, was achieved through competition for winter food supplies. In a similar way, the wildebeest (*Connochaetes taurinus*) population in the Serengeti increased from a level (250 000) at which it was held by the Rinderpest disease in the 1950s to a new level of 1.3 million in 1977 at which it levelled out [Fig. 7.5(a)]. This example is less satisfactory than the elk example for we do not know what the pre-Rinderpest density was, but evidence on reproduction and condition does suggest Rinderpest held the population below the level of the food supply, a necessary condition to demonstrate regulation. There is however, direct evidence [Fig. 7.5(b)] that the

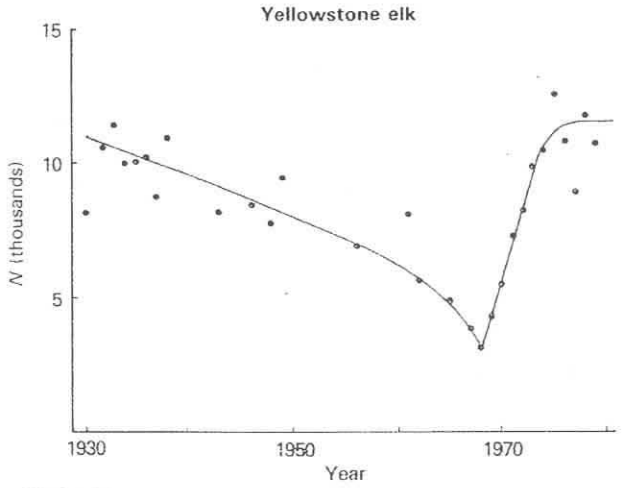

FIG. 7.4 Perturbation of the northern yellowstone elk (*Cervus elephus*) population by culling which stopped in 1968. The population has returned to approximately pre-culling levels. [After Houston (1982).]

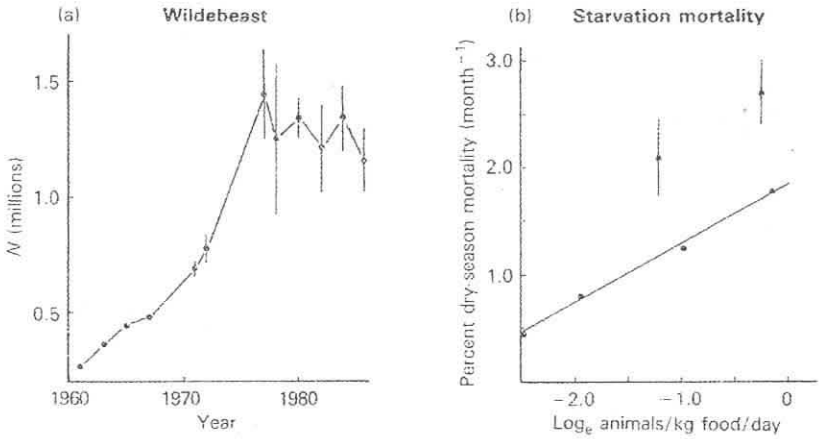

FIG. 7.5 (a) Increase and levelling off of the wildebeest (*Connochaetes taurinus*) population in Serengeti after the virus disease Rinderpest, died out in 1963. Vertical lines are one standard error.
(b) Density-dependent dry season mortality caused by lack of food as the population increased (partly from Sinclair, Dublin & Borner (1985) and from unpublished data). Regression line for the years 1968–72 was used to predict the slope of the line for the two points 1982–83. The slope remained the same, although absolute mortality was higher. Vertical lines are 95% confidence limits.

mechanism for regulation was through density-dependent dry season mortality through lack of food (Sinclair, Dublin & Borner 1985).

Another even less common approach to detecting regulation is a method discussed by Reddingius (1971). The existence of a relationship between an external factor and density, measured from separate populations not influenced by migration, indicates that regulation is taking place (Fig. 7.6), since in an environment exhibiting random variations, this effect on mortality and reproduction will also vary randomly, sometimes resulting in greater mortality than reproduction and sometimes the reverse. Although any one population can remain within relatively narrow limits without regulating factors playing a part, it is extremely improbable that several independent populations can do this and also show mean densities that are correlated with some environmental factor. Some examples include African finches (Schluter 1988), thrips (Klomp 1962) and African buffalo (Sinclair 1977).

By far the most abundant evidence for regulation comes from measures of density dependence. It should be recognized, however, that: (i) density dependence is often overlooked (Gaston & Lawton 1987); (ii) the identification of density dependence may not be sufficient to account for the observed regulation, for there may be other undetected density-dependent factors; and (iii) density-dependent effects may not invariably lead to dampening oscillations, as some factors may have effects leading to stable limit cycles or increasing fluctuations, as seen in some predator–prey models (May *et al.* (1974). This third caveat applies more to insect–parasitoid interactions than to vertebrate populations. On the whole, the detection of density dependence is a less satisfactory form of evidence for the regulation of populations.

Density dependence in insect populations has been reviewed by Solomon (1964), Podoler & Rogers (1975), Stubbs (1977), Dempster (1983) and Strong, Lawton & Southwood (1984) amongst others. Examples of such relationships are shown in Fig. 7.3. Dempster (1983) considered that as much as one third of Lepidoptera studies failed to detect density dependence, but as mentioned above, this proportion may be too high. Nevertheless, in thirty-one studies covering a wide array of insects, Strong, Lawton & Southwood (1984) record eleven with no identifiable density dependence, although subsequently in one, the viburnum whitefly, density dependence has been reported (Hassell *et al.* 1987). In general, the occurrence of density dependence is the rule and I suspect that as census techniques improve, more examples will be reported.

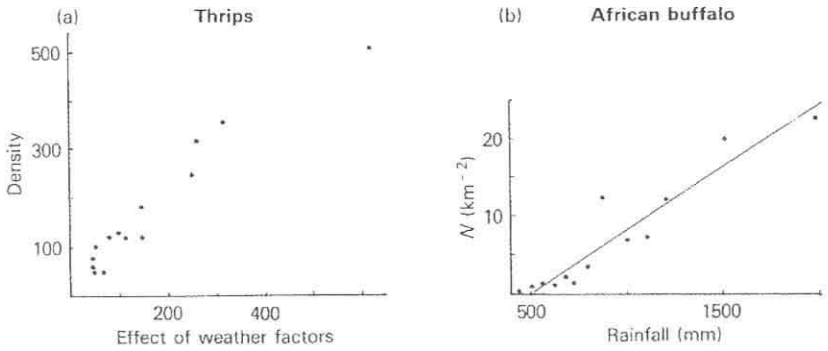

FIG. 7.6 (a) Geometric mean density per rose of thrips (*Thrips imaginis*) from separate populations plotted against an index of weather, as evidence for regulation. [Klomp (1962) after Andrewartha & Birch (1954).]
(b) Mean density of African buffalo (*Syncerus caffer*) from separate populations in East Africa as a function of rainfall, which determines the amount of grass food. [After Sinclair (1977).]

Amongst vertebrates, there are fewer data perhaps because of the long-term records that are necessary. For fish populations there are few attempts to measure density dependence directly, and the work of Elliott (1987) on trout stands out as he demonstrated density dependence both in first winter, and in summer survival [Fig. 7.7(a)]. On the other hand, Doherty (1983) found no evidence for density dependence in the territorial responses of damsel fish. Evidence of regulation in other fish populations, particularly marine fishes, and in marine invertebrates (molluscs, crustaceans), comes indirectly from Ricker-type stock-recruitment curves (Larkin 1973). This evidence is sufficiently strong to warrant mention. Ricker plots show reproductive offspring versus parent stock, the data describing a convex curve if regulation occurs. These curves can be shallow with weak regulation or domed with strong regulation but if the dome is too high, cyclicity or instability occurs. The literature contains many examples of stock-recruitment curves implying regulation (see reviews in Cushing 1981; Parrish 1973). Cushing & Harris (1973) found evidence of regulation in all thirty-one fish stocks they examined, ranging from relatively weak in Atlantic herring and Pacific salmon, intermediate in flatfish, to strong in gadoid fishes. Elasmobranchs show weak to intermediate degrees of regulation (Holden 1973). In molluscs there is evidence of regulation in cockles (*Cardium edule*) and perhaps in the Pacific razor clam (*Siliqua patula*), but for oysters,

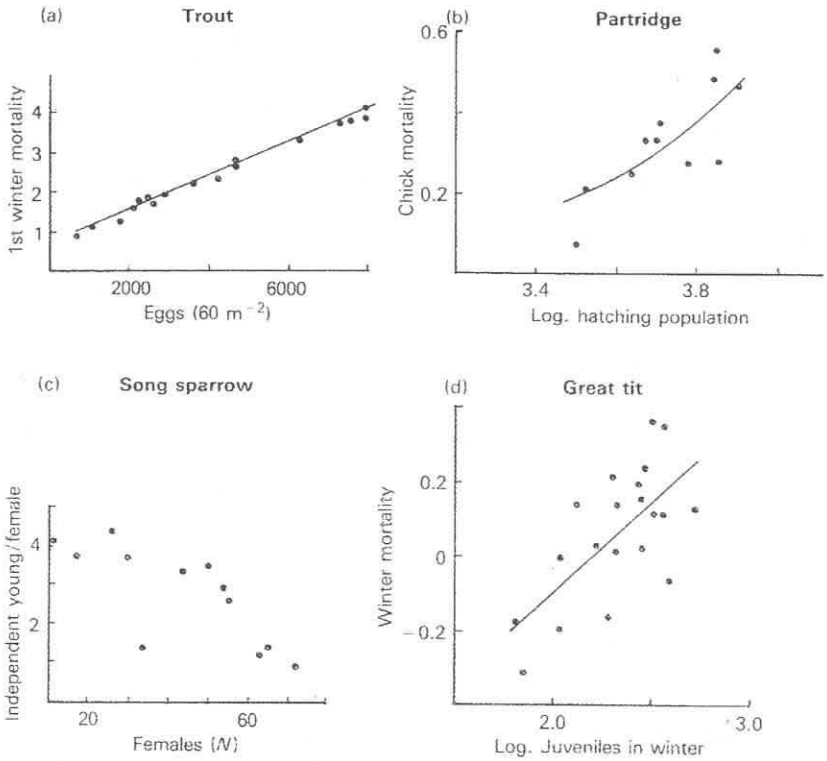

FIG. 7.7 Examples of density dependence in some vertebrate populations.
(a) Trout (*Salmo trutta*) first winter mortality. [After Elliott (1987).]
(b) Partridge (*Perdix perdix*) chick mortality as log hatching population/log population at 6 weeks on a Hampshire estate. [After Blank, Southwood & Cross (1967).]
(c) Song sparrow (*Melospiza melodia*, independent young per female in autumn on Mandarte Island, British Columbia. [After Arcese & Smith (1988).]
(d) *k*-value for winter mortality (log juveniles in winter/first year breeding population) of great tits (*Parus major*) against log juvenile density in winter at Wytham Wood, Oxford. [After McCleery & Perrins (1985).]

mussels and scallops the density-independent effects of temperature and other factors obscure any possible stock-recruitment relationship (Hancock 1973). For crustacea, stock-recruitment curves show evidence of regulation in some cases, such as crabs (Jamieson 1986), lobsters (Addison 1986; Caputi & Brown 1986; Fogarty & Idoine 1986) and prawns (Penn & Caputi 1985), but in many other cases, recruitment was highly influenced by occasional good year classes surviving because of environmental conditions and consequently regulation was not detected.

Finally, for aquatic and marine zooplankton there has been little attempt to find evidence of regulation. The reasons for this are probably the difficulty of measuring single populations for stock-recruitment or life-cycle census data, although Kerfoot, DeMott & DeAngelis (1985) have measured density dependence in *Daphnia* reproduction. Nevertheless, plankton populations are particularly suited to perturbation experiments and these would seem a profitable and exciting area for further research.

Before 1975, there were few studies on birds which showed density dependence, those of Perrins (1965), Krebs (1970) and Kluyver (1971) on great tits, Southern (1970) on tawny owls, and Blank, Southwood & Cross (1967) [Fig. 7.1(b)] on partridges stand out. In the 1980s there has been an increase in reports of density dependence, for example, in pied and collared flycatchers (Stenning, Harvey & Campbell 1988; Torok & Toth, 1988) and song sparrows (Arcese & Smith 1988) [Fig 7.7(c)]. Some of the early great tit data have now been re-examined with longer records. For the years 1947–1961 Perrins (1965) found that clutch size declined as the number of breeding pairs increased, but the data for the period 1962–1983 show no relationship (McCleery & Perrins 1985). Thus there are temporal changes in density dependence, a feature also noted by Stenning, Harvey & Campbell (1988) but which has not previously been considered important in population studies. The present great tit data show that winter mortality is now density dependent [Fig. 7.7(d)] whereas previously it was hardly detectable (Krebs 1970). The greater sample sizes are showing up new, or confirming previously less robust density-dependent relationships (see also Tinbergen, Van Balen & Van Eck 1985). Nevertheless, even with relatively long data records, 17 years for pied flycatchers (Stenning, Harvey & Campbell 1988) and 22 years for great tits in Holland (Tinbergen, Van Balen & Van Eck 1985), the density-dependent effects can be weak and hard to detect.

Regulation of mammal populations is better known for large mammals (see Fig. 7.8 and reviews by Fowler 1981, 1987; Fowler & Smith 1981) than for small mammals. With the latter group, work has been concerned with limiting factors and causes of cycles, rather than with a search for density dependence. Studies on large mammals are often confounded by human interference through commercial harvesting, yet such harvesting can provide the right conditions for a perturbation experiment. Fowler (1987) reports over 100 studies of separate populations of terrestrial and marine large mammals where density dependence has been detected. It is not yet possible to give an estimate of the prevalence of density dependence but I suspect it will be found in most cases. Fowler (1987) notes that density-dependent mortality is non-linear

with density, the strongest effects occurring at high density [resembling the high densities of Fig. 7.1(b)].

Stubbs (1977) has suggested that animals living in temporary habitats (the *r* strategists) tend to suffer high, overcompensating and destabilizing density-dependent effects due to scramble competition; all the competitors use the limiting resource until it begins to run out and then only a few individuals survive. Here the density dependence is curvilinear. Animals in permanent habitats (*K* strategists) have exact or undercompensating mortalities due to interference competition; this allows some of the competitors to obtain sufficient resource to reproduce while others obtain very little and die. Fowler (1981) however, concluded that large mammals (*K* strategists) show most density-dependent change at high population levels, while insects, fish and similar *r* strategists show most density-dependent changes at low population levels. Fowler's conclusion may not contradict that of Stubbs because most of the *r*-type species that Fowler refers to could be regulated by predators rather than by competition (Strong, Lawton & Southwood 1984). Stubbs (1977) refers mostly to those *r* strategists which experience intraspecific competition. Nevertheless, the pattern of density dependence in its relation to life-history strategies is still vague. This is an area for further analysis.

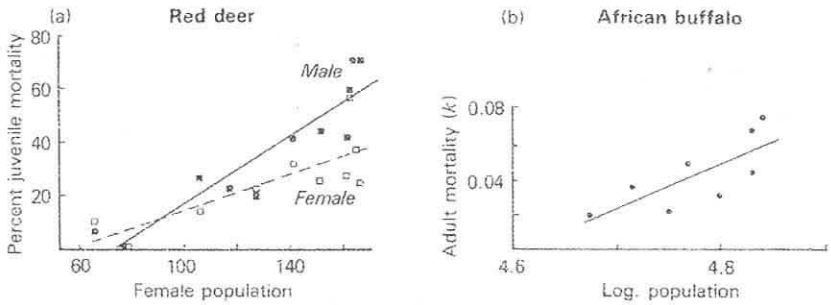

FIG. 7.8 Density dependence in large mammals.
(a) Mortality of female and male juvenile red deer (*Cervus elephus*, on Rhum. [After Clutton-Brock, Major & Guinness (1985).]
(b) *k*-value of adult mortality for African buffalo. [After Sinclair (1977).]

DENSITY DEPENDENCE IN THE LIFE CYCLE

I have examined which stages in the life cycle are subject to regulatory influences, and I have compared these stages between animal groups. Data were obtained from the reviews of Cushing & Harris (1973), Stubbs (1977), Fowler (1981, 1987), Strong, Lawton & Southwood (1984),

McLaren & Smith (1985), and a number of subsequent reports (Table 7.1). I have divided the studies into insects, fish, birds, small mammals (largely rodents), large marine mammals (seals, whales) and large terrestial mammals, which include ungulates, carnivores and primates.

Life stages examined for insects are eggs, larvae (early juvenile), pupae (late juvenile) and adults. Out of the forty-seven case studies, most reported density dependence occurred at the larval stage. Fecundity or egg production was the next most common stage for density dependence and only a few reported regulation in adult stages. For marine fishes, Cushing (1981) found that the degreee of regulation acting on a population was directly related to the fecundity of the species; thus gadoids with high fecundity experienced strong regulation, while salmonids with relatively low fecundity showed weak regulation, and elasmobranchs with the lowest fecundity showed even weaker regulation (Holden 1973). Nevertheless, it was the early larval stages of the life cycle where density dependence occurred in the thirty one stocks examined by Cushing & Harris (1973), as it was for trout (*Salmo trutta*) (Elliott 1987) and Pacific sardine (Iles 1973). However, Le Cren (1987) found that fertility was density dependent in perch (*Perca fluviatilis*) and this was suggested for elasmobranchs (Holden 1973). In general, fisheries biologists assume that juvenile mortality is density dependent while adult mortality is density independent (Charnov 1986).

Similarly in marine crustaceans, density-dependent mortality was assumed to occur in juvenile prawns (Penn & Caputi 1985), crabs (Jamieson 1986) and western rock lobster (*Panulirus longipes*) (Chittleborough & Philips 1975). However, there may be density-dependent mortality of the larger adult lobsters in some species because they cannot find sufficient shelters (Addison 1986).

In contrast to insects and fish, 74% of the bird studies reported density-dependent mortality in the later juvenile stages which included independent young during their first summer and winter, and in two studies included adult mortality as well. There are some reports of clutch size and early juvenile (nestling, fledging) mortality being density dependent but these were less frequent. Overall 56% of studies reported density dependence in the breeding season (eggs to fledgings) and 61% in the non-breeding season (autumn recruitment and winter mortality). Thus the data are split and do not unequivocally support either of the hypotheses of Lack (1966) or Wynne-Edwards (1962). Since all of these studies are in temperate regions, these data may well be biased, as processes in the tropics could be acting on earlier life stages (e.g. nestling mortality through predation). This is an area for new work.

Few small mammal studies address the question of regulation. All

TABLE 7.1. Number (%) of reports of separate populations demonstrating density dependence at different life stages

Group	Fertility/ egg prodn *N* (%)	Early juvenile mortality *N* (%)	Late juvenile mortality *N* (%)	Adult mortality *N* (%)	Total no. populations
Insects[1]	14 (30)	19 (40)	13 (28)	6 (13)	47
Fish[2]	2 (6)	33 (94)	0	0	35
Birds[3]	5 (26)	6 (32)	14 (74)	4 (21)	19
Small[4] mammals	0	0	12 (92)	1 (8)	13
Large[5] marine mammals	34 (83)	10 (24)	0	1 (2)	41
Large[6] terrestrial mammals	49 (68)	35 (49)	1 (1)	12 (17)	72

[1] From Clark *et al.* 1967; Gage, Miller & Mook 1970; Solomon *et al.* 1976; Stubbs 1977; Kenmore *et al.* 1984; Strong, Lawton & Southwood 1984; Chambers, Wellings & Dixon 1985; Jones *et al.* 1987; Hill 1988; Roland 1988; Southwood & Reader 1988.
[2] From Cushing & Harris 1973; Holden 1973; Iles 1973; Elliott 1987; Le Cren 1987.
[3] From Blank, Southwood & Cross 1967; Southern 1970; Ryder 1975; Goss-Custard 1980; Zwickel 1980; Hannon, Sopuck & Zwickel 1982; Alatalo & Lundberg 1984; Ekman 1984; Hill 1984; Greig-Smith 1985; Hudson, Dobson & Newborn 1985; McCleery & Perrins 1985; Tinbergen *et al.* 1985; Newton & Marquiss 1986; Nilsson 1987; Porter & Coulson 1987; Arcese & Smith 1988; Stenning, Harvey & Campbell 1988; Torok & Toth 1988.
[4] From Healey 1967; Jannett 1978; Cockburn 1981; Hestbeck 1982, 1987; Boonstra & Rodd 1983; Galindo & Krebs 1987; Trostel *et al.* 1987.
[5] From Fowler 1981, 1987; Fowler & Smith 1981.
[6] From Fedigan 1983; Skogland 1985; Fowler 1987; Kruuk & Parish 1987.

eleven rodent studies concluded that there was density-dependent exclusion of late juveniles into the breeding population prior to breeding. In contrast, a study on snowshoe hares (*Lepus americanus*) (Trostel *et al.* 1987) has recorded adult mortality overwinter as the possible regulating stage. In large marine mammals, almost the opposite result is found; either female fertility (all whale species reported and some seals) or early juvenile mortality (suckling seal pups) were density dependent. Large terrestrial mammals also reflect the effects of density-dependent factors on fertility and juvenile mortality but a few studies record density dependence in winter (or dry season) mortality of late juveniles and adults.

On the face of it, there are large differences between groups in where regulation occurs during the life cycle. However, there are inherent biases

in the way species are studied which distort the distribution. Thus in marine mammals, almost nothing is known about late juvenile and adult mortality because it takes place at sea. Similarly, little is known about fertility changes and nestling mortality of small animals because of the difficulty of measuring these life stages. For large terrestrial mammals, few studies have attempted to measure adult mortality directly so this stage is likely to be under-represented as it probably is in birds. Thus generalities must remain tentative and future research must concentrate on obtaining a more complete picture of mortality during the life cycle.

Stubbs (1977) concluded that most of the density-dependent mortality in the life cycle acts on the young stages for species living in temporary habitats, while it acts more on fecundity in those species living in permanent habitats. With the present data one can modify this. Species with very high reproductive rates (insects, fish) have early juvenile (eggs, larvae) density-dependent mortality; those with intermediate reproductive rates (birds, small mammals) have late juvenile and prebreeding regulation, while large mammals with low reproductive rates are at least partly regulated through changes in fertility. I suspect this is an incomplete picture and in large mammals at least, more cases of late juvenile density dependence will be found. As noted earlier from Fig. 7.1 the earlier in the life cycle that regulation takes place, the greater the sensitivity the population shows to changes in regulation and hence, one sees greater fluctuations in population size for species with early life-cycle regulation.

CAUSES OF DENSITY-DEPENDENT MORTALITY

A large number of studies have considered the causes of mortality that limit populations. As discussed above, this is a relatively uninformative exercise unless one also knows whether the mortality is density-dependent or not. The *k*-factor analysis of Varley & Gradwell (1968) can provide information on 'key factors', those mortalities which account for most of the total population change from year to year. These can be either density dependent or density independent (Podoler & Rogers 1975) and it is important to identify the causes of key-factor mortality. However, in studies of vertebrate populations even this analysis is rare, so the results of most studies are of limited usefulness. Many, but not all cases were from those referenced in Table 7.1. In addition, some reports describe causes without reference to life stage. Table 7.2 presents the frequency of case histories where the cause of density-dependent mortality was identified.

TABLE 7.2. Number (%) of reports of separate populations recording cause of density dependence. Population totals differ from those in Table 7.1 because some studies report causes but not the life stage for density dependence, and other studies the reverse. For insects, parasites include parasitoids

Group	Space *N* (%)	Food *N* (%)	Predators *N* (%)	Parasites *N* (%)	Disease *N* (%)	Social *N* (%)	Total no. popu-lations
Insects	0	23 (45)	20 (39)	19 (37)	5 (10)	4 (8)	51
Large marine mammals	0	6 (60)	4 (40)	0	0	0	10
Large terrestrial mammals	1 (1)	71 (99)	0	0	2 (3)	0	72
Small mammals	14 (67)	5 (24)	4 (19)	0	0	14 (67)	21
Birds	5 (33)	8 (53)	0	1 (6)	0	7 (47)	15

Sources as for Table 7.1 but in addition, for insects Faeth & Simberloff 1981; Jones 1987; for small mammals other sources in Erlinge *et al.* 1983; Hestbeck 1987; for marine mammals McLaren & Smith 1985; for large terrestrial mammals Sinclair 1977.

Insects

The insects provide an interesting paradox. On the one hand, Table 7.2 indicates that from fifty-one studies, 45% of the populations were at least partly regulated through competition for food causing either larval mortality or reduced fecundity; a conclusion also reached by Dempster (1983) for Lepidoptera. On the other hand, some insect ecologists (e.g. Myers 1987; Price 1987) point to the strong indirect evidence for predator–parasitoid regulation: (i) biological control experiments where introduced pests are regulated by their natural predators, or where weeds are not controlled by introduced insect herbivores because the latter are regulated by predators; (ii) the use of pesticides which release pests by killing their natural predators. For example, the brown planthopper (*Nilaparvata lugens*) is a pest of rice plants and is regulated by spiders causing density-dependent nymphal mortality. Application of insecticide killed the spiders and caused an 800-fold increase in brown planthopper densities (Kenmore *et al.* 1984); and (iii) insect antipredator defences can confer a 30% or greater survival advantage over individuals without the defence (Price 1987). The same life-table data used by Dempster (1983) were analysed by Strong, Lawton & Southwood (1984) and they reached the opposite conclusion, namely that predation was the most frequent cause of regulation. It would appear that life-table analyses are applied

mostly to pest species whose populations are known to erupt and reach food limitation, thus they are a biased sample of insect populations and we know little about most insect species that do not noticeably affect their food supply. Moreover, this ignorance lies at the centre of the debate on whether predation or interspecific competition structures ecological communities.

The effect of diseases caused by viruses, bacteria and fungi has been largely overlooked in field studies, but their significance as a cause in the collapse of insect outbreaks (Anderson & May 1980) and in the production of insect population cycles is becoming more apparent (Ewald 1987; Myers 1988). The few cases involving 'social' causes of regulation cited emigration of adults due to crowding. It is clearly not a common form of regulation but it is seen in aphids, locusts and planthoppers.

Fish

Causes of density-dependent mortality in fish stocks are so little known that they have not been included in Table 7.2. It is generally considered that there is an interaction between food supplies and predators affecting early larval stages. High density leads to competition and lack of food which causes slow growth and hence prolongs the stage where fish are vulnerable to predators such as birds, other fish species or even adults of their own species (e.g. Jones 1973; Cushing 1981; Lasker 1985; Le Cren 1987). In essence fish show density-dependent growth.

Large mammals

In marine mammals the predominant cause of regulation has been assumed to be food supplies affecting fertility and growth of juveniles. Recently McLaren & Smith (1985) have suggested that predators on seals such as polar bears, arctic foxes, leopard seals and sharks, may be regulating the populations of several prey species.

For large terrestrial mammals, the overwhelming cause of regulation in Table 7.2 is food supply. Many of these studies involved direct measures of food (e.g. for wildebeest, Sinclair, Dublin & Borner 1985) but in other studies, food is inferred from effects on fertility. In contrast to this, there is a general perception amongst North American wildlife ecologists that large mammal herbivores are regulated by predators (Keith 1974; Bergerud, Wyett & Snider 1983; Bergerud 1988). Unfortunately, no studies demonstrate the density-dependent effects of predation,

most contenting themselves with the observation that populations increase when predators are removed. This rather predictable result merely allows the trivial conclusion that predators are limiting their prey populations but it does not imply regulation. In Africa, there is some circumstantial evidence for regulation of prey populations by predators, for example resident antelopes in the Serengeti Park by predators in general (Sinclair 1985) and wildebeest by lions in the Kruger Park (Smuts 1978), but conclusive evidence is lacking. The absence of critical evidence for density-dependent effects by predators highlights the lack of appropriate questions and research programmes in wildlife ecology.

To demonstrate regulation of prey by predators from a predator removal experiment, it is not enough to show an increase in the prey population. Regulation is demonstrated only if: (i) the prey population increases when predators are removed; and (ii) the prey population does not return to its original density after predators have been allowed to return to their previous levels. This can be illustrated by reference to Fig. 7.1(c) where a prey population moves from K_3 (predator regulated) to K_1 then back to K_2 (predator limited but not regulated). In any other case (initial prey population at K_4 or K_2) the removal experiment produces the same qualitative behaviour and therefore, ambiguous results; in these cases k-factor analysis is needed in addition. So far none of these conditions has been met. Large mammal populations may well be regulated by predators but this has not yet been demonstrated.

Small mammals

In fourteen of the twenty-one small mammal studies (Table 7.2) the important cause of regulation was density-dependent exclusion of juveniles from breeding colonies, and presumably this exclusion was due to competition for space. Lidicker (1975) considered that in the microtine rodents which show population cycles, juveniles emigrate voluntarily in increasing populations before resources become limiting. However, the difficulties of measuring resources for rodents are great and it remains open whether or not emigrants are detecting early signs of food depletion and using this as a cue to leave (which would mean regulation by interference competition for food). Various food addition experiments (e.g. Taitt & Krebs 1981; Ims 1987) show that rodent populations are monitoring their food supplies, and Ostfeld (1985) considered that food supplies are the underlying cause of territoriality in microtines.

Predation is implicated in the regulation of populations of three species, field voles (*Microtus agrestis*), wood mice (*Apodemus sylvaticus*) and snowshoe hares (Keith *et al.* 1977; Erlinge *et al.* 1983; Trostel *et al.*

1987). In all cases, predators acted in a delayed density-dependent way (Fig. 7.9). For voles and mice in southern Sweden, the delayed action of predators (mainly foxes and buzzards) was within the annual cycle. Erlinge *et al.* (1983) argued that heavy predation acted to dampen the 4–5 year cycles which occur in northern Sweden. However, Kidd & Lewis (1987) have shown that these predators act, if anything, in an inverse density-dependent way and are thus destabilizing; they concluded that these predators are merely limiting and not regulating. Trostel *et al.* (1987) reported the delayed action of raptors (great horned owls, goshawks) and carnivores (lynx, coyotes) causing winter mortality on snowshoe hares, and suggested through a simulation model, that this process could produce the 10-year cycle of numbers.

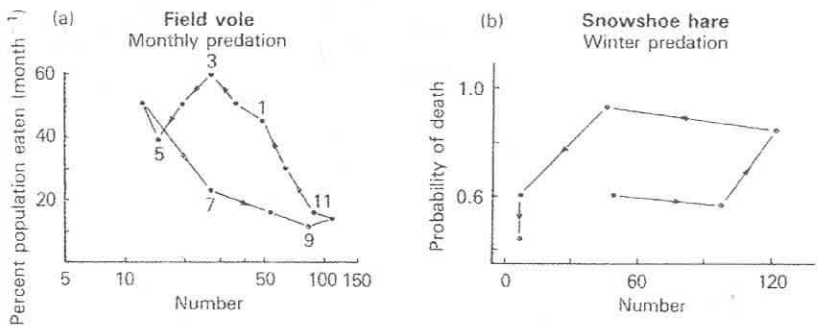

FIG. 7.9 Delayed density-dependent predation on small mammals, indicated by an anti-clockwise spiral when points are joined in temporal sequence.
(a) 'Monthly predation mortality of field voles (*Microtus agrestis*). [After Erlinge *et al.* (1983).]
(b) Annual predation mortality in winter on snowshoe hares (*Lepus americanus*). [After Trostel *et al.* (1987).]

In most studies of small mammals, predation has not been measured. Indirect evidence, for example from the provision of cover (Taitt *et al.* 1981), showed that predation by birds could be important in causing the decline phase in vole cycles and suggests more attention should be paid to this aspect.

Birds

Predation is noticeably absent as a cause of regulation in bird populations although it is suggested as a limiting factor in some gallinaceous and grouse species (Newton 1980). Many studies have recorded the whole life

cycle and it is unlikely that predation has been overlooked. These studies have, however, been in temperate regions and it is quite possible that predation could be a regulating process in the tropics.

Lack (1966) proposed that bird populations were limited by food shortage. Despite much uncritical and circumstantial evidence concerning the role of food supply, there seems to be support for Lack's view that food is an ultimate limiting factor (Newton 1980). Whether food shortage acts as a direct regulating factor is not so clear. Table 7.2 shows that foods shortage was the cause in 53% of the cases where density-dependence was recorded, while competition for territorial space or nesting sites accounted for 33% of the cases.

The cases where space is regulating are included as 'social' causes of regulation together with feeding interference (Goss-Custard 1980). Territoriality has been cited as a cause of regulation, particularly in grouse populations (Watson 1967; Zwickel 1980; Hannon, Sopuck & Zwickel 1982). The implication is that territoriality prevents some birds breeding (demonstrated by removal of breeding birds and observing others taking over) and thereby depresses recruitment in a density-dependent way. This predicts an increasing proportion of non-breeders as density increases, and has been demonstrated in song sparrows (Fig. 7.10). If all those without territories die, as in red grouse (Watson & Moss 1980; Watson 1985), then one can conclude that competition for territorial space is regulating. If food addition experiments show that territory decreases in size, (Watson, Moss & Parr 1984) then, as suggested by Lack (1966), the regulating factor is competition for food, and territoriality (or other forms of social behaviour) is the secondary, proximate mechanism which determines who obtains the food. In this case, territoriality (i.e. competition for space) is not a regulating factor. Therefore, both food and territoriality are limiting factors (Watson & Moss 1970; Goss-Custard 1980) but not necessarily regulating factors. Food and spacing behaviour may alternate in their effects depending on density (Boag & Schroeder 1987). Parasites (helminths) and spacing behaviour may act synergistically to regulate red grouse populations, and parasites may be producing cycles in grouse numbers (Hudson, Dobson Newborn 1985) in a way reminiscent of diseases acting on insect populations (Anderson & May 1980).

In many studies, it is concluded that territoriality regulates breeding numbers (Brown 1969; Patterson 1980) but often there is little knowledge about the non-breeding population. Without information on the non-territory holders, one cannot conclude that territorial behaviour regulates the population, for there could be a compensating density-

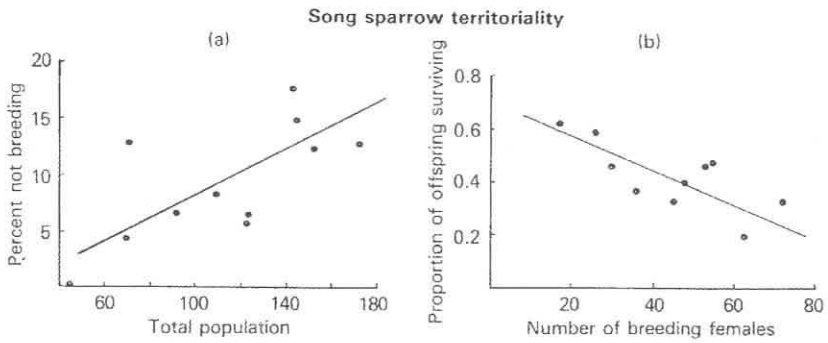

FIG. 7.10 Two periods of density dependence during the year for song sparrows. (Data from P. Arcese & J. N. M. Smith, personal communication.)
(a) Proportion of total population (males plus females) on Mandarte Island, British Columbia, excluded from breeding. During the breeding season territoriality excludes an increasing proportion as density increases.
(b) Proportion of offspring surviving in the non-breeding season declines with density of females.

dependent mortality either amongst the non-territory holders or in the non-breeding season (Hannon & Zwickel 1987). For example, in the song sparrow territoriality produces a density-dependent proportion of non-breeders (Fig. 7.10) (P. Arcese & J. N. M. Smith, personal communication), but there is also a density-dependent mortality in the non-breeding season which is equally effective in regulating the population. A food addition experiment allowed many non-breeders to become territory holders. Thus, much of the regulation took place before breeding and territoriality merely determined who could breed (Arcese & Smith 1988). A similar situation has been reported for mallards (Hill 1984). In summary, before regulation can be attributed to spacing behaviour, it is necessary to measure the mortality of birds in the non-breeding season; many of the studies claiming that territoriality of breeding birds regulates a population are, therefore, premature.

To conclude this section on causes of regulation, are there any clear generalities? At present the answer is no, partly because the picture is incomplete. Both predation and resource limitation are operating in some groups. Nevertheless predation has not been studied adequately in any animal group and the effects of disease and parasitism have been almost entirely neglected. The importance of epidemiological studies is now being recognized, but far more basic information is required to develop these aspects.

NON-EQUILIBRIUM CONDITIONS, MULTIPLE STABLE STATES AND THE WAY AHEAD

The high variance in numbers in many natural populations has challenged ecologists to explain the persistence of such populations (Connell & Sousa 1983; Schoener 1985; Ostfeld 1988). It is recognized that unregulated hypothetical populations describe random walks over time and eventually go extinct (Reddingius 1971). Since all species do go extinct, it is possible that present day populations are merely those following a random walk in transition to extinction. Strong (1984) argues that extinctions in random walk models are far too frequent to mimic the behaviour of real populations. However, there is no way that this statement can be tested. The strength of the regulation argument is that it predicts the existence of a process (density dependence) which can be measured in nature. The philosophical weakness of the argument is that it is very hard to reject; absence of density dependence can too easily be explained away on *ad hoc* methodological grounds.

The original alternative hypothesis to regulation was that of Andrewartha & Birch (1954): namely that density-independent factors changed sufficiently frequently that at least some sub-populations remained extant, and were able to recolonize other areas where sub-populations had become extinct. They considered that this process of chance events is particularly important at the edge of a species' range. The idea of many sub-populations recolonizing local extinctions and resulting in persistence without regulation was later developed more fully (e.g. Reddingius & Den Boer 1970). Although the idea continues to attract some workers (Ehrlich *et al.* 1972), the weight of evidence is in favour of the regulation hypothesis.

Nicholson (1958) recognized that where the environment fluctuates, populations may become extinct and are later reestablished; in these situations the regulatory factors would be relatively weak and masked by density-independent factors. Such a scenario can be illustrated by Fig. 7.11 which shows how stochasticity in the density-independent mortality results in greater changes in population size when the density-dependent mortality is weak than when it is strong. These ideas have been rephrased in recent years under the heading of 'density -vague regulation' by which Strong (1984, 1986) suggested there is stochasticity in both the density-dependent as well as the density-independent variables. In addition Strong (1984) stated that '... regulation at extremes of density can involve very different mechanisms from mechanisms around central or average densities'. Since average densities tend to occur in the centre of a species' range, this is not saying much more than that originally

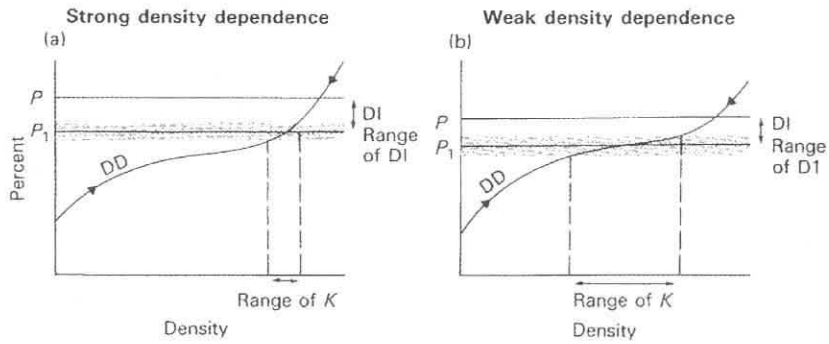

FIG. 7.11 Effect of stochasticity in the density-independent mortality (DI), the range of which is indicated by the shaded area, results in less fluctuation in the equilibrium K, when the density-dependent mortality (*DD*) is strong (A) than when it is weak (B).

conceived by Nicholson, and which is illustrated by the curvilinear density dependence in Fig. 7.1(b). Although different causes of regulation may operate at the centre and edge of a species' distribution (e.g. Randall (1982) described how the moth, *Coleophora alticolella*, is regulated at low altitude by predators, mid-altitude by competition for food, and at high altitude is subjected to fluctuation and local extinction by climate), such differences in mechanisms are not a necessary condition for curvilinear density dependence. In short, density vague phenomena may simply be caused by curvilinear density dependence so that at some densities regulation is strong, at others weak (e.g. Whittaker 1971; Elliott 1987).

Nevertheless, it is quite conceivable that multiple equilibria exist for a population [Fig 7.1(c)]. The ideas concerning multiple stable states (Holling 1973; May 1977) can be applied to both ecosystems and populations. Indirect evidence for multiple equilibria comes from studies on the spruce budworm (*Choristoneura fumiferana*) (Peterman, Clark & Holling 1979) [Fig. 7.12(a)] and other forest insects (McNamee, McLeod & Holling 1981; Berryman, Stenseth & Isaev 1987), salmonids (Peterman, Clark & Holling 1979), marine fish stocks and plankton (Steele & Henderson 1981, 1984), and wildebeest (Walker & Noy-Meir 1982). Many of these examples suggest regulation by predators at lower densities and by competition for food at higher densities. Escape from lower stable points could occur through a variety of mechanisms; for example, through the growth of forest food supply for the spruce budworm (Peterman, Clark & Holling 1979), rapid immigration in other forest insects (Berryman, Stenseth & Isaev 1987), and by climatic effects on the grass food of wildebeest. In addition the existence of multiple equilibria can depend on the behaviour of a population. For example,

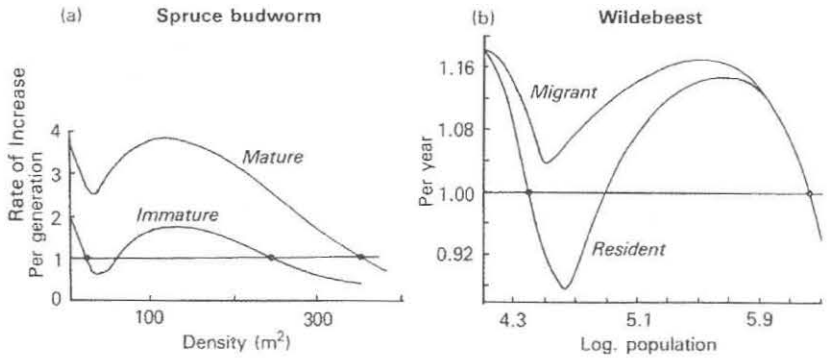

FIG. 7.12 Examples of possible multiple stable states, where the ratio N_{t+1}/N_t is plotted against density.
(a) Spruce budworm in immature conifer forests has two stable points (•). As the forest matures the lower point disappears, (redrawn from Peterman, Clark & Holling 1979). (b) Wildebeest resident populations could have two stable points, whereas migrant populations have only one. Modelled for the 25 000 km² Serengeti ecosystem, Tanzania. [After Fryxell, Greever & Sinclair (1988).]

migratory wildebeest in Serengeti [Fig. 7.12(B)] have only one upper equilibrium point, set by food supply as they possibly cannot be regulated by predators. In contrast, resident populations have two stable points and may be regulated by predators (Fryxell, Greever & Sinclair 1988). Hestbeck (1982, 1987) has suggested that small mammal populations can be regulated at different levels by spacing behaviour, predation and food supply.

These examples remain largely hypothetical interpretations of indirect evidence. Although there is some direct evidence of multiple stable states in jack pine sawfly (*Neodiprion swainei*) (McLeod 1979; Holling 1986) evidence on the causes of these equilibria is almost entirely lacking. There are also alternative interpretations of the spruce budworm data suggesting that there is no predator stability point (Royama 1984). The concept of multiple equilibria requires field testing both by field perturbation experiments (see above) or by measures of density dependence. The existence of multiple equilibria predicts: (i) that there are two (or more) densities where there is strong density dependence; (ii) that some of the percentage mortality at lower density should be higher than some of that at higher density, i.e. the two k-factors should overlap; and consequently (iii) there should be an inverse density-dependent effect at an intermediate density preceding a direct density-dependent effect at higher density.

These predictions have not yet been explicitly tested, but there is one example which has superficially similar phenomena. Stenning, Harvey &

Campbell (1988) showed for pied flycatchers (Fig. 7.13) two mortalities k_1, k_4 which conform to predictions (i) and (ii), and a third mortality k_3, which conforms to prediction (iii). True agreement with these predictions requires that there should be different underlying causes for the different slopes.

So far I have discussed multiple causes of regulation in terms of sequential effects at different densities. It is possible, however, that two

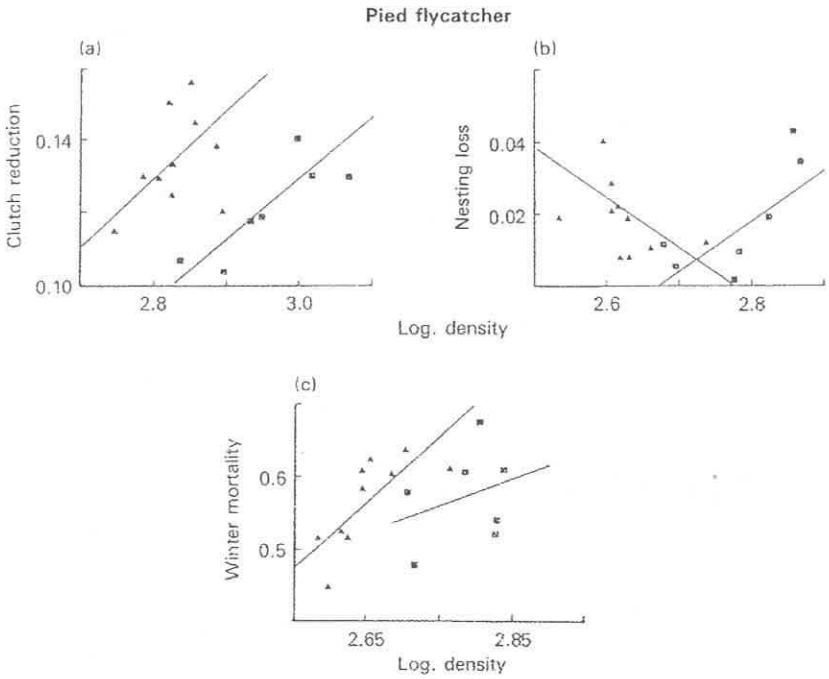

FIG. 7.13 Possible evidence for multiple stable states in the pied flycatcher (*Ficedula hypoleuca*) comes from changes in mortality in the time periods 1948–54. (triangles) and 1955–64 (squares). Density for 25 ha in the Forest of Dean, Gloucestershire. [After Stenning, Harvey & Campbell (1988).]
(a) k-value for clutch size reduction shows two densities where there is strong density dependence.
(b) k-value for mortality of nestlings shows inverse density dependence at lower densities and positive density dependence at higher densities.
(c) k-value for winter mortality also shows two densities with positive density dependence.

or more causes may be operating simultaneously and synergistically. This is an area of work that deserves far more attention, but some recent examples are pointing the way ahead. The introduced winter moth (*Operophtera brumata*) in Nova Scotia was originally thought to be

controlled by introduced parasitoids. Recent re-analysis of the data has shown that pupal mortality is the regulating stage and this mortality is caused by beetle larvae but only after parasitoid introduction; in effect parasitized pupae grow slower and are thus more vulnerable to predation (Roland 1988). A similar interactive effect of predators and parasites is reported by Jones (1987), where larvae of the butterfly *Pieris rapae*, which were parasitized by braconid parasites, then became vulnerable to ant predation. Essentially parasitism slowed growth (analogous to the density-dependent growth caused by lack of food in many fish populations) and resulted in density-dependent predation. In other invertebrate species, including cockroaches, amphipods, isopods and ostracods, Moore (1984) demonstrated how acanthocephalan parasites alter the behaviour of hosts so that they become more vulnerable to predators and Curtis (1987) described how trematodes alter snail behaviour. Hudson, Dobson & Newborn (1985) have proposed an interaction of parasitic worms and behaviour as the regulating process in red grouse , and an interaction of disease and food could regulate some ungulate populations (Sinclair 1977). Predation not only mitigates competition but it can also promote it. Mittelbach & Chesson (1987) showed how predators concentrate small size classes of several species into a common protective habitat, thereby intensifying competition.

Apart from studies of fish populations, a few other examples suggest an interaction between food and predators; in snowshoe hares, one hypothesis proposes a simultaneous effect of food shortage and predation causing the decline phase of the 10-year cycle (Trostel *et al.* 1987). This predicts that at high density, animals that run out of food are not only slower to avoid predators but also expose themselves longer to predation; essentially this is density-dependent antipredator behaviour. In a remarkable series of studies, Goss-Custard and co-workers (e.g. Goss-Custard & Durell 1987; Selman & Goss-Custard 1988) have shown how shore bird populations may be regulated in winter through the interaction of feeding behaviour, age and social status, and food supply. McNamara & Houston (1987) have modelled the interaction of feeding and antipredator behaviour in terms of the individual; we now need to incorporate the effects of population density in the models.

CONCLUSIONS

Perhaps the first thing that is needed in future work is a more rigorous attention to terminology to avoid the continuing confusion of meaning in the literature. A survey of the literature has shown that we still have a

poor understanding of where density dependence occurs in the life cycle of almost every group of animals. There are very few studies in the tropics so far, a major gap since it is likely that dynamic processes there could be very different from those in temperate regions; our knowledge of regulation in marine and aquatic life cycles is also minimal.

Further work is needed on the causes of density dependence in the rarer insect species, nearly all fish populations and in the effects of predation on birds and mammals. We need to pay attention to single or multiple causes of regulation, and if multiple, how the various factors interact, for example whether sequentially, synergistically or antagonistically.

Studies are beginning to show that regulation is not always constant. Long-term studies indicate temporal changes in the strength of regulation and other dynamic behaviour in the same population, while other studies have recorded spatial differences in regulation. For the most part, we know nothing about the underlying causes of these changes; are they local or global, short or long term? Are they due to qualitative differences in habitat and species composition or to quantitative shifts in variables? McCauley & Murdoch (1987) provide one approach for *Daphnia* but we have hardly started to explore these aspects. They are important from the point of view of biological control, for we need to know the conditions when control could operate and when it might break down.

There is still a paucity of good perturbation experiments, both to demonstrate regulation and to understand its mechanism. Aquatic species are difficult subjects for the detection of density dependence, but equally they are very suitable for perturbation experiments; progress could be made in this area.

Despite these gaps in our knowledge we have advanced since the 1957 Coldspring Harbor meeting. Unlike that symposium we now have a general model that accommodates all the current ideas on regulation — curvilinear, delayed and inverse density dependence, stochastic effects, and multiple stable states. Understanding of regulation may have been slow, but we should recognize that it takes a long time. We now have a considerable body of evidence demonstrating regulation in many groups; in 1957 there was almost none. Furthermore our ideas have become more sophisticated leading to a recent resurgence of interest in the dynamic behaviour of populations.

In reviewing the history of ideas on regulation, one sees how theories evolve. Theories have not been discarded because of a critical test or series of tests, but rather they wither away either because they are untestable [e.g. Thompson's (1939) multifactor hypothesis] or not

heuristic, or there is lack of evidence in their support (e.g. group selection), while others expand because of more evidence in their favour or their power to generate new ideas. Nicholson's theories of equilibrium led in different ways to the works of both Wynne-Edwards (1962) and Lack (1966) and to the outburst of field studies. It is perhaps not appreciated that by 1957 Nicholson had anticipated most of the modern concepts on regulation; local extinctions, curvilinear density dependence and density-vague phenomena.

Perhaps the most interesting and challenging area for future work lies in a better understanding of both the mechanisms and causes producing multiple stable states. At present, we have only circumstantial evidence for these phenomena. This aspect could well be the most important advance we make in population dynamics in the next few decades. Such an understanding is vital not only for the management of ecosystems on a regional scale but also for the understanding of global changes that are now confronting us in stark reality, such as the change in atmospheric carbon dioxide and the ozone layer, the decline of tropical forests, the spread of aridity, the human population explosion in Africa. These are all examples which may well lead to complex, non-linear reponses in populations and ecosystems and which we must be able to predict.

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